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Southwestern MEDICINE

Official Journal of The Southwestern Medical Association, The Western Association of Railway Surgeons, The Texas Orthopaedic Association, The Southwest Obstetrical and Gynecological Society, The Southwestern Dermatological Society, Texas District One Medical Association, The Southwestern New Mexico Medical Society, and El Paso County Medical Society

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April, 1960

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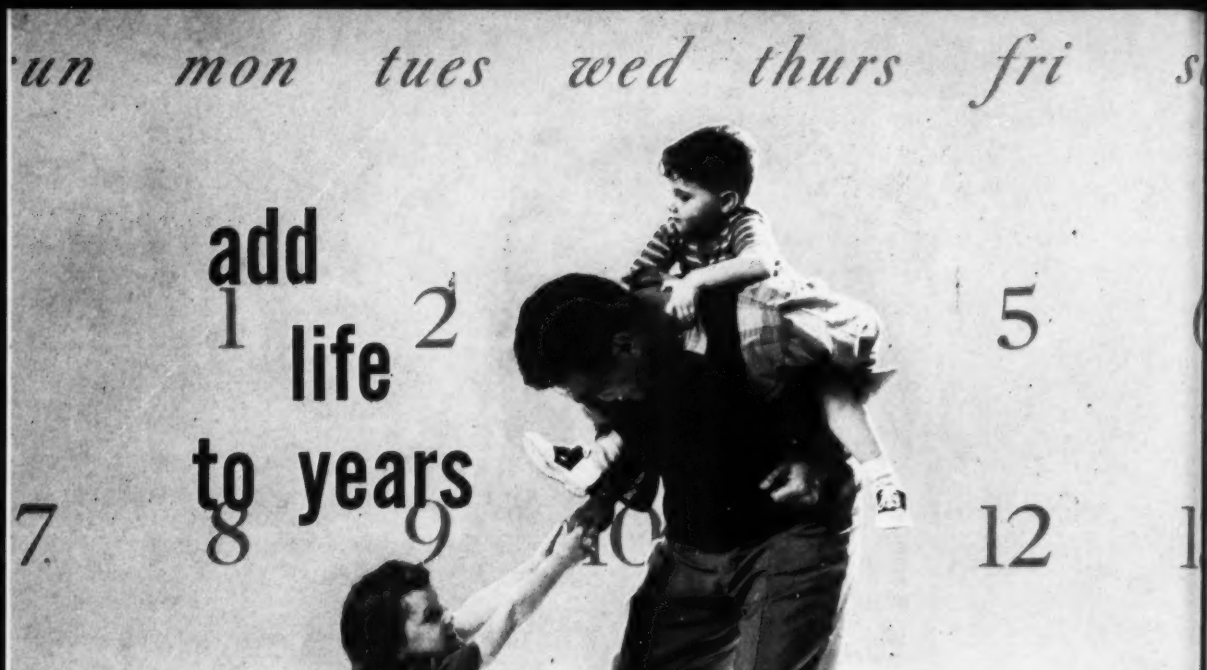

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1. Spies, T. D.: The Influence of Nutritional Processes on Aging, South. M. J., *50*:216, 1957.

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Coming Meetings

Texas Orthopaedic Association, Annual Meeting, Hilton Hotel, Fort Worth, April 11, 1960.

Arizona Medical Association, 69th Annual Meeting, Safari Hotel, Scottsdale, Arizona, May 4-7, 1960.

New Mexico Medical Society, Annual Meeting, Western Skies Hotel, Albuquerque, May 8-14, 1960.

Postgraduate Course, Dermatology for General Practitioners, University of Colorado Medical Center, Denver, July 14-16, 1960.

Postgraduate Medical Assembly of South Texas, Shamrock Hilton, Houston, July 18-20, 1960.

Postgraduate Course in Pediatrics, University of Colorado School of Medicine, The Stanley Hotel, Estes Park, Colorado, Sept. 1-6, 1960.

Southwestern Medical Association, 42nd Annual Meeting, Hotel Hilton, El Paso, Oct. 20-22, 1960.

Southwest Obstetrical and Gynecological Society, Annual Meeting, Riviera Hotel, Las Vegas, Nev., Nov. 7-8, 1960.

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SOUTHWESTERN MEDICINE

VOL. XLI, NO. 4 APRIL, 1960

New Mexico Medical Society to Hold 78th Annual Meeting in May in Albuquerque

Plans have been completed for the 78th annual meeting of the New Mexico Medical Society, May 8 to 14, in the Western Skies Hotel in Albuquerque, N. M.

Speakers will be Dr. Schuyler G. Kohl, Brooklyn, N. Y., Associate Professor of Obstetrics and Gynecology and Assistant Dean at the State University of New York's Downstate Medical Center; Dr. Bob Robins, Camden, Ark., member of the Board of Trustees, American Medical Association; Dr. Richard L. Day, Pittsburgh, Professor of Pediatrics at the University of Pittsburgh Medical School; Dr. Jerome Hilger, Minneapolis, Professor of ENT at the University of Minnesota Medical School; Dr. David Post, Albuquerque; Dr. Randolph Seligman, Albuquerque; Dr. Roy Goddard, Albuquerque, Director of Pediatric Research for the Lovelace Foundation for Medical Education and Research;

More Speakers

Dr. Jerome Hilger, Minneapolis; Dr. Albert W. Egenhofer, Santa Fe; Dr. Jack Mosier, Albuquerque; Dr. Joseph L. Hollander, Philadelphia, Professor of Medicine at the University of Pennsylvania Medical School; Dr. John L. Otto, Galveston, Professor of Neuro-Psychiatry at the University of Texas Medical Branch at Galveston; Dr. Frank Krusen, Professor of Physical Medicine and Rehabilitation at the Mayo Foundation and Special Assistant for Health and Medical Affairs to the Director of Vocational Rehabilitation, Department of Health, Education and Welfare;

Dr. Denton Cooley, Houston, Associate Professor of Surgery at Baylor University Medical School; Dr. John R. Betson, Albuquerque, Lovelace Clinic; Dr. Louis H. Winer, Los Angeles, Clinical Professor of Dermatology at the University of California at Los Angeles; Dr. Benson Bloom, Albuquerque, Director of Professional Services at the Veterans Administration Hospital; Dr. Guy E. Rader, Albuquerque; Dr. John C. Murphy, Albuquerque, Chief of the Dermatological Section at the Lovelace Clinic;

Still More Speakers

Dr. Winslow Bashe, Columbus, Ohio, Director of Communicable Disease Division of the Ohio State Health Department; and Dr. John Colbeck, Vancouver, B. C., Chief of Service on Pathology at Shaughnessy Hospital.

Officers of the Society are Dr. Lewis M. Overton, Albuquerque, President; Dr. Allan L. Haynes, Clovis, President-Elect; Dr. William Badger, Hobbs, Vice-President; and Dr. Thomas L. Carr, Albuquerque, Secretary-Treasurer.

Program

Sunday, May 8
Council Meeting, Territorial Room
Monday, May 9

8:30 A.M. First Indoctrination Course for New Members
La Mina Room

MEETINGS

Tuesday, May 10

8:30 A.M. House of Delegates
La Mina Room

6:30 P.M. Cocktail Party for Officers, Delegates, Speakers and their Wives

7:30 P.M. Banquet for Officers, Delegates, Speakers and Wives
Address: Bob Robins, M.D., Camden, Ark., American Medical Association Board of Trustees, "The New Look in Medical Practice"

Wednesday, May 11
General Meeting
Sandia Room

Presiding: James C. Sedgwick, M.D., Immediate Past President, N. M. Medical Society

8:45 A.M. Call to Order
Invocation: Paul Calhoun, Pastor, Immanuel Presbyterian Church, Albuquerque
Welcome: Maurice Sanchez, Chairman Albuquerque City Commission
Welcome: Omar Legant, M.D., President Bernalillo County Medical Association

9:00 A.M. Presidential Address:
Lewis M. Overton, M.D., Albuquerque, President, New Mexico Medical Society

General Scientific Session No. 1
Sandia Room

Presiding: Lewis M. Overton, M.D.
Co-Chairman: Earl B. Flanagan, Jr., M.D., Carlsbad

9:30 A.M. "New York Cerebral Palsy Study"
Schuyler G. Kohl, M.D., Associate Professor Ob-Gyn, and Assistant Dean, State University of New York, Downstate Medical Center, Brooklyn

10:00 A.M. "Bilirubin and Jaundice Mechanisms in Infants"
Richard L. Day, M.D., Professor of Pediatrics, University of Pittsburgh Medical School, Pittsburgh

10:30-11:00 Recess to Visit Exhibits

11:00 A.M. To Be Announced

11:30 A.M. "Surgical Treatment of Hearing Loss"
Jerome Hilger, M.D., Prof. ENT, University of Minnesota Medical School, Minneapolis

12:00 Noon Specialty Luncheons as Arranged

Group Scientific Session A
La Mina Room

Session A-1—Problems of the Newborn

Presiding: Alvina Looram, M.D., Santa Fe, Director Child Health Division, Department of Public Health

Co-Chairman: Howard Smith, M.D., Roswell, President N. M. Obstetrical-Gynecological Society

2:00 P.M. "The Maternal and Infant Mortality Committee of the New Mexico Medical Society"
David Post, M.D., Albuquerque

2:30 P.M. "Obstetrical Aspects of the Problem in New Mexico"
Randolph V. Seligman, M.D., Albuquerque, Chairman, Section

Dr. Cooley

Dr. Day

Dr. Kohl

on Obstetrics, Maternal and Infant Mortality Committee

3:00 P.M. "Pediatric Aspects of the Problem in New Mexico"
Roy Goddard, M.D., Albuquerque, Director, Pediatric Research, Lovelace Foundation for Medical Education and Research

3:30 P.M. Afternoon Break
Symposium: "The Improvement of Perinatal Morbidity and Mortality"
Presiding: Stuart W. Adler, M.D., Albuquerque, President, New Mexico Pediatric Society

3:45 P.M. "An Evaluation of Obstetrical & Newborn Services in the Community"
Schuyler G. Kohl, M.D., Brooklyn

4:15 P.M. "Respiratory Problems of Premature & Newborn Infants"
Richard L. Day, M.D., Pittsburgh

4:45 P.M. To Be Announced

5:15 P.M. Suggestions for Improvement in Maternal-Infant Mortality in New Mexico

Session A-2—Problems of the Special Senses
Sandia Room

Presiding: Jack Dillahunt, M.D., Albuquerque, President, New Mexico Ophthalmological Society
Co-Chairman: George Richardson, M.D., Roswell

2:00 P.M. "Tinnitus"
Jerome Hilger, M.D., Minneapolis

2:30 P.M. "External Diseases & Anterior Segment of the Eye"
Albert W. Egenhofer, M.D., Santa Fe

3:00 P.M. "Aphasia, Apraxia and Agnosia"
Jack Mosier, M.D., Albuquerque

3:30 P.M. Afternoon Break

3:45- 5:00 Panel: Discusson on Special Senses
Moderator: Arthur J. Fischer, M.D., Las Cruces
Discussants: Drs. Hilger, Egenhofer and Mosier

6:30 P.M. Cocktail Hour for All Registrants

7:30 P.M. Specialty Group Dinners
New Mexico Pediatric Society
New Mexico Radiology and Pathology Society
New Mexico Obstetrics and Gynecology Society

Thursday, May 12
General Scientific Session No. 2
Sandia Room

Presiding: Allan Haynes, M.D., Clovis, President-Elect, N. M. Medical Society
Co-Chairman: Andrew Babey, M.D., Las Cruces

8:45 A.M. Call to Order and Announcements

9:00 A.M. "Present Day Concepts in the Management of Rheumatoid Arthritis"
Joseph L. Hollander, M.D., Professor of Medicine, University of Pennsylvania Medical School, Philadelphia

9:30 A.M. "Uses and Abuses of Tranquilizers in Chronic Illnesses"
John L. Otto, M.D., Professor of Neuro-Psychiatry, University of Texas, Galveston

Dr. Krusen

Dr. Otto

Dr. Robins

10:00 A.M. "Rehabilitation Adds Life to Years"
Frank Krusen, M.D., Professor of Physical Medicine and Rehabilitation, Mayo Foundation, Special Assistant for Health and Medical Affairs to the Director of Vocational Rehabilitation, Department of Health, Education and Welfare

10:30 A.M. Recess to Visit Exhibits

11:00 A.M. to 12:30 P.M. Panel Discussion: Rehabilitation
Moderator: Lewis M. Overton, M.D.
Discussants: Drs. Hollander, Krusen and Otto

Afternoon **Free**

6:30 P.M. Cocktails—Four Hills Country Club

7:30 P.M. Dinner Dance (Social, no speeches)

Friday, May 13
General Scientific Session No. 3
Sandia Room

Presiding: W. E. Badger, M.D., Hobbs, Vice-President New Mexico Medical Society

Co-Chairman: R. C. Derbyshire, M.D., Santa Fe, President, New Mexico Section, American College of Surgeons

8:45 A.M. Call to Order and Announcements

9:00 A.M. "Open Heart Surgery"
Denton Cooley, M.D., Associate Professor of Surgery, Baylor University Medical School, Houston

9:30 A.M. "Cancer in Pregnancy"
John R. Betson, M.D., Lovelace Clinic, Albuquerque

10:00 A.M. "Tumors of the Skin, Benign and Malignant"
Louis H. Winer, M.D., Clinical Professor Dermatology, University of California at Los Angeles

10:30 A.M. Recess to Visit Exhibits

11:30 A.M. to 12:00 Noon Panel Discussion: "Cancer of the Breast"
Moderator: Martin Goodwin, M.D., Clovis, Secretary, N. M. Radiology & Pathology Society
Discussants: Drs. Cooley, Kohl, Winer, Otto

12:15- 1:45 P.M. Specialty Group Luncheons

Group Scientific Session B
Blue Room

Session B-1—Cardio-Pulmonary Problems in the Aged

Chairman: Carl H. Gellenthien, M.D., Valmora, President, N. M. Chapter, American College of Chest Physicians

Co-Chairman: Quentin J. Florence, M.D., Roswell, President, New Mexico Heart Association

2:00 P.M. "Pulmonary Problems of the Aged"
Benson Bloom, M.D., Director, Professional Services, V.A. Hospital, Albuquerque

2:30 P.M. "Cardiovascular Problems in the Aged"
Denton Cooley, M.D.

3:00 P.M. "Non-Visceral Chest Problems in the Aged"
Joseph L. Hollander, M.D.

3:30 P.M. Afternoon Break

3:45- 5:00 Panel Discussion: "Crippling Diseases of the Chest"
Moderator: Burgess Gordon, M.D., Albuquerque, Past President, American College of Chest Physicians
Discussants: Drs. Cooley, Hollander, Krusen & Otto

Session B-2—Problems of the Skin
La Mina Room

Presiding: George A. Waldriff, M.D., Albuquerque, President, Southwestern & Rocky Mountain Dermatological Societies

Co-Chairman: Earl Pace, M.D., Santa Fe

2:00 P.M. "Skin Problems in Pediatrics"
Guy E. Rader, M.D., Albuquerque

2:30 P.M. "Pigmented Lesions of the Skin"
Louis H. Winer, M.D.

3:00 P.M. "Allergic Skin Problems"
John C. Murphy, M.D., Albuquerque, Chief, Dermatological Section Lovelace Clinic

3:30 P.M. Afternoon Break

3:45- 5:00 P.M. Panel Discussion: "Skin Problems"
Moderator: John J. Corcoram, M.D., Albuquerque

6:30 P.M. Open for Individual Cocktail Parties

7:30 P.M. Specialty Group Dinners
New Mexico Chapter, American College of Chest Physicians

Saturday, May 14

Symposium on Infectious Disease Control—La Mina Room
(Co-sponsored by the Public Health Committee of the New Mexico Medical Society & New Mexico Department of Public Health)

Co-Chairman: Roy F. Goddard, M.D., Chairman Public Health Committee, N. M. Medical Society

Co-Chairman: Stanley Leland, M.D., Santa Fe, Director, N. M. Department of Public Health

9:00 A.M. "The Problem of Infectious Disease Control"
Winslow Bashe, M.D., Director, Communicable Disease Division,, Ohio State Health Department, Columbus

9:45 A.M. "Hospital Programs"
John Colbeck, M.D., Chief of Pathology Service, Shaughnessy Hospital, D.V.A., Vancouver, British Columbia

10:30 A.M. Coffee Break

10:45 A.M. Movie: "Hospital Sepsis — A Communicable Disease"

12:15- 1:45 P.M. Luncheon

2:00 P.M. Panel: "The Public Health Team"
Moderator: John Mason, Santa Fe, D.V.M., M.P.H., Chief, Infectious Disease Division, N. M. Department of Public Health

3:00 P.M. Discussion: "What Can Be Done in New Mexico?"
Moderator: Howard L. Wilson, M.D., Los Alamos

4:00 P.M. Resume and Recommendations
Drs. Bashe and Colbeck

WOMEN'S AUXILIARY

Program

Tuesday, May 10

3:00 to 5:00 P.M. Coffee for State Officers, County Officers and Committee Chairmen
Suite 139: Hostess, Mrs. Martin Goodwin, President, Auxiliary to the New Mexico Medical Society

Wednesday, May 11

8:45 A.M. Opening Ceremonies of New Mexico Medical Society
Sandia Room

9:45 to 11:45 A.M. House of Delegates Meeting
Territorial Room

12:00 A.M. Luncheon — Pool Side
Speaker: American Medical Association Auxiliary Representative

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Dr. Loraine Orr Dutton

Dr. Loraine Orr Dutton, 61, El Paso, who came to El Paso in 1931, died Jan. 23, 1960.

He was a member of the American College of Allergists, American Academy of Allergy, Texas Society of Pathologists, American Assn. of Blood Banks, director of the clinical laboratories of Providence Memorial Hospital and R. E. Thomason General Hospital, and medical director of the Southwest Blood Bank in El Paso. He was a Past President of the Southwest Allergy Forum.

Dr. Neidich of Las Cruces Elected President of Southwestern New Mexico Medical Society

Dr. E. K. Neidich of Las Cruces, was elected president of the Southwestern New Mexico Medical Society at its annual meeting in Las Cruces, January 18.

Other new officers were Dr. Russell Lane, Silver City, vice-president; and Dr. William J. Hossley, Deming, secretary. The retiring president was Dr. Paul A. Feil of Deming.

Speaking at the meeting, which attracted 85 physicians and wives from southern New Mexico and El Paso County, was Harold J. Crasilneck, Ph.D., of Dallas, assistant professor of psychiatry at the University of Texas Southwestern Medical School in Dallas. Dr. Crasilneck spoke on "The Uses and Abuses of Hypnotism." He was presented an award by the Society for Clinical and Experimental Hypnosis in 1958 for writing the best paper in the world on hypnotism.

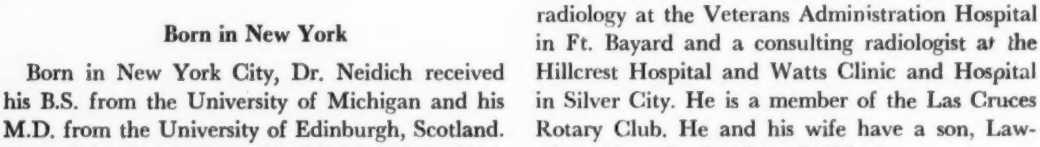
Born in New York

Born in New York City, Dr. Neidich received his B.S. from the University of Michigan and his M.D. from the University of Edinburgh, Scotland. He took his internship and residency in New York City and in 1941 began the general practice of medicine in Devonshire, England. He entered the Army in 1941 as a major and served with the Army Medical Corps from 1941 to 1946 with four years overseas duty in the European and North African Theatres of war. He emerged from the service with the rank of major.

Between 1947 and 1951 he did residency work in radiology in New York City and became a Diplomate of the American Board of Radiology in 1951. He began the practice of radiology that year in New York City and in 1953 moved to Las Cruces, New Mexico, where he is now a practicing radiologist.

He is a member of the American College of Radiology, the American Roentgen Ray Society and a past president of the Dona Ana County Medical Society. He has been a consultant in radiology at the Veterans Administration Hospital in Ft. Bayard and a consulting radiologist at the Hillcrest Hospital and Watts Clinic and Hospital in Silver City. He is a member of the Las Cruces Rotary Club. He and his wife have a son, Lawrence, 11, and a daughter, Judith, 6.

SOUTHWESTERN NEW MEXICO OFFICERS—Participating in the annual meeting of the Southwestern New Mexico Medical Society at Las Cruces, January 18, were, left to right, Dr. William J. Hossley, Deming, secretary; Dr. Russell Lane, Silver City, vice-president; Harold J. Crasilneck, Ph.D., Dallas, speaker; Dr. Paul A. Feil, Deming, retiring president; and Dr. E. K. Neidich, Las Cruces, new president. Dr. Crasilneck, who is assistant professor of psychiatry at the University of Texas Southwestern Medical School, Dallas, spoke on "The Uses and Abuses of Hypnosis."



THE PRESIDENT'S PAGE

Young Men of Medicine

(Continued)

By RUSSELL L. DETER, M.D., *El Paso*
President, Southwestern Medical Association

In the last issue we discussed briefly our responsibility to the high school student who wants to study medicine, and the pre-medical student.

In this issue, we shall attempt to cover briefly the part we should play in the education, training and preparation of young doctors to practice medicine.

Taking my topics in order —

3. The Medical Student, Intern and Resident.

We should think of all three of these as different phases of medical education per se. The medical student is taking large intellectual bites of the science of medicine and is beginning to acquire a life long habit of self-initiated learning. In this phase we, as practitioners (to differentiate from full time professors), do not play too active a part. We should take every opportunity to enjoy their enthusiasm, visit with them, and regain some of that vitality so characterized by the enthusiastic medical student.

We must help them explore the magnificent new vistas opening up for them as they learn anatomy, physiology, etc., ad infinitum. We should never, at this optimum stage of training and development, bring up some of the unpleasant relations which sometimes exist between doctor and patient, or doctor and other doctors, and the transitory trials and tribulations of organized medicine. There is too much for them to assimilate without interjecting these unpleasant facts at this stage.

Medicine Should Be Fun

We should, as practitioners, emphasize and teach them that medicine should be fun and should be exciting; and, if it isn't, they are probably in the wrong field. We should, at this level, also impress on them that they should choose the specialty that they enjoy the most, not the one which might be the most remunerative. Whether it be in teaching, research or practice is of no importance, if they like what they are doing. Teach them that the young man who starts out to make money as a primary objective seldom does, but the young man whose primary objective is to render service can hardly help but make an adequate income.

Dr. Deter

They should be taught, somewhere along the way, that if they do not want to work they shouldn't hire out. It is pointless to study medicine and take intensive graduate training, and then go out and only take calls during day-time hours, none on Saturday or Sunday, and always be difficult to find. A doctor has only one commodity to sell and that is himself and his knowledge of medicine—he must go and be where his services are needed, and that is with his sick patient. Patients can be trying in their demands, but they **are sick.**

Same Philosophy

The intern and resident must be exposed to much of this same philosophy, but in addition they must also be taught the art of medicine and that is at the patient's bedside, in the operating room and in the out-patient clinic.

We must get away from the idea that interns and residents are there to take over some of the unpleasant duties of patient care, writing up charts, etc. The reason you have an intern and resident program is to turn out better trained doctors of medicine.

This goal cannot be achieved without your being there to give him the benefit of your experience. The intern or resident is not there to take over the care of indigents, he is there to learn how to become a better doctor of medicine.

4. The Young Doctor Starting Out in Practice.

We must lend him a helping hand when he gets started in practice and try to warn him of the pitfalls of professional relations with other doctors. We must help him in setting up fees commensurate with those being charged in our community.

Above all else, do not discourage him in new fields, or unexplored fields, in medicine. These are the young men who approach the mass of unsolved problems in medicine with that enthusiasm which comes from not knowing that all of these problems are insoluble. Every day some young man in medicine is finding a solution to these so-called "insoluble" problems. Because you have not achieved your goals, or solved the many problems of medicine, does not mean that they cannot be solved.

If you can't help in these projects, don't be a wet blanket. Console them in their failures, praise them their successes.

It's Time We Reevaluate Our Public Relations Program

By Sol Heinemann, M.D., *Carlsbad, N. M.*

Our present program of public relations is a program of expediency. It should be redefined and recognized for what it is, or else it will merely soften our changeover into socialized medicine. We have been trying for the past 10 years to give people a "good" opinion of physicians as a group. With each new problem that has been tossed at the medical profession, we have sought to soften the blow by good public relations, and solve the problem of the moment with the most expedient solution.

This solution is usually one that has ignored the principle involved, and has usually led us one step closer to putting the practice of medicine in the hands of bureaucracy. In short, we have been trying to come to terms with socialism and are moving closer and closer to socialism. It has been the experience of those who have worked on public relations programs that we have not done this well. Our present public relations programs themselves have not been too successful, inasmuch as our members are "too busy" to devote adequate time to a good public relations program.

Strategic Retreat

It becomes evident that private medicine can only survive under a system of Democracy that supports capitalism. We must decide if we should continue our strategic retreat with delaying actions,

or accept our duty as citizens in defending a capitalist society. As individuals we can only do the latter by becoming actively interested in politics and economics, as here is where the decisions are made.

At the risk of appearing trite, I would like to point out that the Magna Carta and the Constitution outlined basic principles for the freedom of the individual. These principles were based upon a system of capitalism; and where in these documents does one find expediency for the problems of the time in which they were written. They did not solve the imminent problems, but did outline the principles.

We should recognize that under the principle of a capitalistic economy we have survived and prospered. Under this same principle there is no use for or need for socialism in the field of medicine or in any other field. All economists today will admit that we are living under a "mixed economy". This is a nice way of saying we are already partly socialized. When one breaks down a socialistic economy, one finds that the government has obtained both political and economic powers. Capital is now under the control of the bureaucracy of government. Under this system medical care, jobs, and the productivity of the economy are dispensed to the individual by the government.

It is for these reasons that I feel that the medical profession should reevaluate its public relations policy. We have successfully refused to accept social security for physicians. While several inroads into our freedoms have occurred, we still are the largest group of people in this country who are the least socialized. It is therefore our duty to devote our efforts to leading this nation back to the principles of the capitalistic society that we profess to believe in. Unless a strong and united effort is made, this nation will continue down the road to socialism and medical care will be only part of the government program.

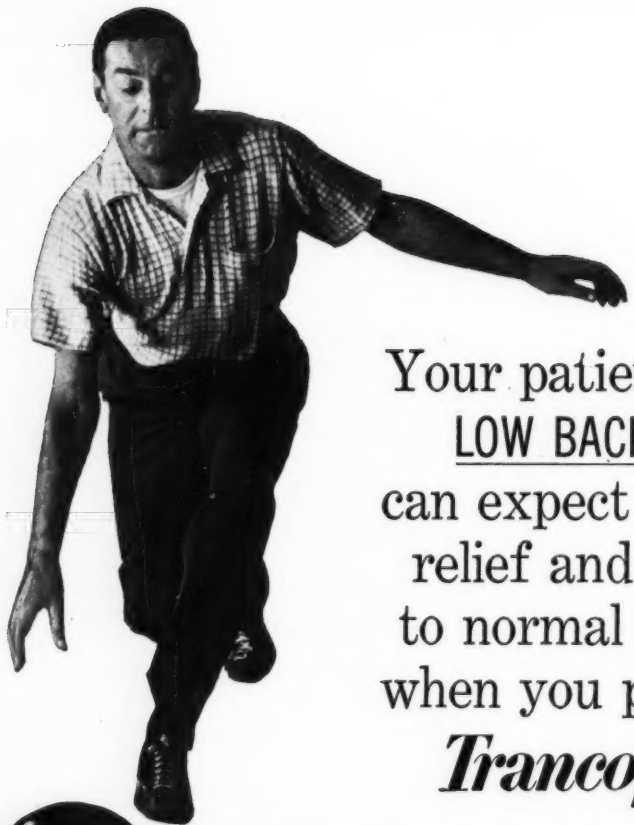
I feel that all doctors should read *The Capitalist Manifesto* by Kelso and Adler, for these authors have grasped the problem and come up with one solution. Their thesis is for capital to be decentralized and placed back into private hands, instead of being centralized in the hands of a bureaucracy.

Where other economists want to experiment with our way of life (all of these experiments have led us into a partially socialized economy) Kelso and Adler would like to reverse this trend. They would like to lead us back to a free competitive economy under the control of the citizens of the nation. One must admit that they have taken an old idea, i.e. capitalism, and are successfully defending its value in the present world, and its worth to the individual.

The basic question then appears to be that if the nation's economic power is in the hands of its citizens, there will be no problem of socialized medicine; but if the economy continues to pass into the hands of the government, socialization is inevitable in all fields. There can be no mixture of the principles of the two.

As a group, doctors should expend their efforts for clarification of the principles involved and furthering those same principles instead of trying to solve the problems as they arise, by the most expedient method. This would involve a strenuous public relations program, but one worthy of our efforts.

As a profession, we can use the power of our public relations department to publicize the problems and to educate the people about the principles involved. Using the A.M.A. as a nucleus, other groups may be induced to act as spearheads to beat a retreat from socialism. This could lead to a revitalization of our nation and might become a rallying point for other nations against communism. Even our allies are finding it difficult to see our differences with communism, when the ship of state is sailing more and more to the left.



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case profile.[1] A 42-year-old truck driver and mover injured his back while moving a piano. The pain radiated from the sacral region down to the region of the Achilles tendon on the right side. X-rays for ruptured disc revealed nothing pertinent. The day of the injury he was given Trancopal immediately after the physical examination. Although 100 to 200 mg. three times a day were prescribed, the patient on his own responsibility increased the dosage of Trancopal to 400 mg. three times a day. This dosage was continued for three days and then gradually reduced over a ten day period. During this time, the patient continued to drive his truck. The muscle spasm was completely controlled and no apparent side effects were noted. For the past six months, the patient has continued to take Trancopal 100 to 200 mg. as needed for muscle spasm, particularly during strenuous days.

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ORIGINAL ARTICLES

The Erythropoietic Hormone in Secondary Anemia.*

By Jack C. Postlewaite, M.D., *El Paso*

The use of cobalt in the treatment of various anemias has been widely studied.[1, 20] It has been shown that the primary effect of cobalt appears in the bone marrow in which the erythroid tissue and the number of mitotic figures are increased.[6, 13, 15] This effect is promptly manifested in the peripheral blood by the appearance of a marked reticulocytosis and subsequent increases in hemoglobin and in the number of erythrocytes.[21, 23]

The mechanism by which cobalt stimulates erythropoiesis has recently been clarified through the work of Goldwasser, Jacobson, Fried and Plzak,[24] who showed that the element increases the production of erythropoietin, the erythropoietic hormone. Cobalt is the only known therapeutic agent having this kind of activity.

Therapeutic administration of cobalt has been confined to the use of the inorganic salt, cobalt chloride. It has proved to be a relatively nontoxic agent in doses of 100 mg. or less per day. Where larger dosage is desirable, however, gastrointestinal side effects and other toxic symptoms have been reported which may preclude its use. This has led to a search for organic cobalt compounds which might prove to be therapeutically superior to the inorganic salt.

The present evaluation was undertaken to determine whether a new organic cobalt complex, Copoietin, was superior to cobalt chloride in clinical practice.

Materials and Methods

Fifty-four patients were treated using Copoietin-Ferrous, a new preparation comprising an organic cobalt complex with ferrous sulfate. Each sugar-coated tablet contained cobalt polyglucopyranose equivalent in cobalt content to the usual 15 mg. dose of cobalt chloride and, in addition, 100 mg. of exsiccated ferrous sulfate. Dosage was one tablet four times daily given with meals and at night before retiring.

There were seven males and 47 females in the series of patients chosen for study. Their ages ranged from 10 to 73 years. These patients were selected chiefly on the basis of presenting symptoms typical of anemia, including weakness, lassitude, fatigue, anorexia and irrtability.

The anemia was characterized as due to iron deficiency of nutritional origin in fifteen patients and resulting from chronic blood loss in an additional six. Twenty-five patients exhibited an anemia associated with infection or with inflammatory disease processes. Included in the series were seven patients having anemia associated with thyroid disease. One patient presented a typical hypoglandular syndrome with mild anemia.

Blood specimens for hematologic examination were obtained at the time of each patient's initial visit, or prior to the initiation of therapy with Copoietin-ferrous, and at each subsequent visit. The interval between visits varied between two weeks and several months, depending upon the patients' cooperativeness. Hemoglobin concentrations were determined photometrically and hematocrit determinations were performed by standard laboratory methods.

Results

The distribution of blood hemoglobin concentrations in 19 patients with iron-deficiency anemia, before and after treatment with Copoietin-ferrous, is shown in Table I. These anemias were characterized by hypochromia and slight microcytosis.

*The compiled statistics above were analyzed and prepared for printing by the research department of Lloyd Brothers, Cincinnati, Ohio.

Two patients were dropped from the series because of failure either to keep appointments or to take the medication according to prescribed directions.

In the remaining group of 19 patients, the average initial hemoglobin concentration was 11.7 ±0.62 grams per 100 cubic centimeters of blood. After treatment for periods ranging from 14 to 141 days, the average hemoglobin concentration had increased approximately 1.6 grams per 100 cubic centimeters to an average level of 13.4 ± 1.03 grams per cent. Before treatment was begun, hematocrits ranged from 34 to 43 per cent, with an average value of 39.0 ± 2.36.

After treatment with Copoietin-ferrous, the average hematocrit had increased to 42.7 ± 2.75 per cent, the range being from 37 to 46 per cent. The mean corpuscular hemoglobin concentrations (M.C.H.C.) of these patients were somewhat low initially, ranging from 27.9 to 32 per cent, with an average of 29.8 per cent.

The values of this index showed no significant change following treatment, ranging from 26.1 to 36.4 per cent, with an average on the low normal side (31.7 per cent).

TABLE I

Patients with Iron-deficiency Anemia

Patient No.	*Etiology*	*Duration of Therapy (days)*	*Hemoglobin (gm./100 cc.)*	
			Initial	*Final*
4742	Nutritional	141	11.2	14.0
764	Nutritional	14	11.7	12.4
337	Nutritional	60	11.7	12.8
4227	Nutritional	31	11.7	14.8
5194*	Chronic bleeding duodenal ulcer	124	12.0	13.0
5344	Menstrual blood loss	49	12.0	13.0
2938	Nutritional	91	11.3	12.4
4260	Nutritional	79	11.7	14.0
421	Nutritional	56	10.0	12.0
5205	Nutritional	20	12.0	12.0
5237	Nutritional	44	12.0	13.0
1595	Chronic bleeding duodenal ulcer	113	12.4	14.0
5239	Nutritional	78	11.2	12.0
128	Nutritional	104	12.4	15.3
3067	Nutritional	119	11.2	13.8
MLN	Menstrual blood loss	108	12.4	14.8
4058*	Nutritional	101	12.8	14.6
BR	Menstrual blood loss	120	11.7	13.0
908	Allergic purpura	69	11.5	12.8

*Male

Table II shows the distribution of blood hemoglobin concentrations, before and after treatment with Copoietin-ferrous, in 20 patients with anemia associated with infection or with inflammatory disease. Five patients were excluded from this series because of failure to keep their appointments or to take the medication as directed.

In the remaining group of twenty patients, before therapy with Copoietin-ferrous was initiated, the blood hemoglobin concentration ranged from 10.6 to 13.1 grams per 100 cubic centimeters, with an average of 12.0 ± 0.81. The hematocrits of these patients varied between 32 and 42 per cent, with an average of 38.6 ± 2.72. These anemias were characterized as normocytic with M.C.H.C. values ranging from 27.5 to 34.1 per cent (av. 31 per cent).

After treatment with Copoietin-ferrous for from 19 to 313 days, these patients' average blood hemoglobin concentration had increased 1.4 grams per 100 cubic centimeters (to 13.4 ± 1.26 gm./100 cc.). Excluding the two patients (4291 and P.S.) who failed to show an improvement, the average increase in blood hemoglobin concentration was 1.6 grams per 100 cubic centimeters.

This was accompanied by an increase in the average hematocrit to 41.5 ± 1.5 per cent (range 39-55 per cent), an average gain of 2.94 per cent. There was a small increase, also, in the average M.C.H.C. value to 32.3 per cent.

In a group of eight patients with anemia and thyroid disease or hypoglandular syndrome (2017), before therapy with Copoietin-ferrous was begun blood hemoglobin concentrations ranged from 8.6 to 12.8 grams, with an average of 11.2 ± 1.2 grams per 100 cubic centimeters. The data relating to these patients are shown in Table III.

TABLE II

Anemia Associated with Infection or Inflammation

Patient No.	*Diagnosis*	*Duration of Therapy (days)*	*Hemoglobin (gm./100 cc.)*	
			Initial	*Final*
5018	Chronic colitis, non-ulcerative	43	12.8	14.0
1248	Infectious mononucleosis	40	12.6	13.3
5152	Hepatitis	126	12.0	13.0
5159	Hepatocholangitis	90	12.0	12.8
515	Hepatitis, colitis	123	11.7	12.8
4811	Chronic pelvic infection	193	11.0	13.6
2347	Endometriosis	127	12.0	13.0
821	Chronic infection	313	13.1	14.8
4841	Amebic colitis	177	13.1	13.8
3651	Gastritis	164	11.7	14.0
100	Chronic tonsillitis	42	10.6	12.6
1086	Post viral pneumonia	309	11.0	13.0
1294*	Bronchitis, allergic and bacterial	68	11.8	17.7
4291	Gallstones	127	12.0	12.0
4609	Rheumatoid arthritis	19	11.0	12.6
5291	Palindromic rheumatism	42	12.8	13.3
5229	Rheumatoid arthritis	28	12.6	14.3
1107	Rheumatoid arthritis	21	12.6	13.1
5054	Rheumatoid arthritis	31	11.0	12.6
PS	Rheumatic chorea	68	12.8	11.7

*Male

TABLE III

Patients with Thyroid Disease

Patient No.	*Diagnosis*	*Duration of Therapy (days)*	*Hemoglobin (gm./100 cc.)*	
			Initial	*Final*
937	Hypothyroid	53	12.0	14.8
5091	Hyperthyroid	140	11.7	11.0
4855*	Hypothyroid	56	12.8	15.5
4708	Thyrotoxicosis	83	10.2	13.1
774	Hyperthyroid	98	8.6	13.0
3198	Hypothyroid	355	11.5	13.1
194	Hypothyroid	121	10.0	12.8
2017	Hypoglandular syndrome	85	12.8	12.6

*Male

Their hematocrits ranged from 25 to 44 percent (av. 37.4 percent) before treatment. In all but two patients the anemia was normocytic and hypochromic. The two exceptional patients exhibited marked microcytosis with M.C.H.C. values in the normal range.

After treatment for periods ranging from 53 to 355 days, six of the eight patients showed marked improvement in their peripheral blood picture. Their blood hemoglobin concentrations had increased by from 1.6 to 4.4 grams per 100 cubic centimeters (av. 2.87 gm./100 cc.), and their hematocrits had increased by from 2.0 to 13.0 percent (av. 5.0 percent). The two patients who failed to show improvement exhibited small decreases in both their hemoglobin and hematocrit values.

In general, this new cobalt complex was extraordinarily well tolerated. Only two patients complained of gastrointestinal distress despite the fact that therapy was continued in some cases for as long as 18 to 45 weeks. After treatment for 17 weeks, one patient developed a rash which disappeared promptly when the medication was stopped. The remaining 51 patients had no side effects and no evidence whatsoever of toxic reaction was observed in any patient.

Discussion

Our series of cases illustrate the two primary types of anemias commonly seen in general practice. The iron-deficiency group is typical in that most of the patients are females in whom the anemia may be "nutritional" in origin or may be thought of as due to lack of sufficient iron absorption to offset menstrual loss. These anemias are hypochromic and sometimes microcytic.

The remainder of the patients had developed an anemia secondary to infection or chronic disease and, in general, showed a normocytic, normochromic anemia except where mild iron deficiency was also present. This combination of factors as a cause of anemia may be more common than has generally been supposed since, in the female, mild to moderate iron-deficiency may already be present when an infection or inflammatory disease is superimposed.

We considered our results in the iron deficiency series to be most satisfactory although no comparison with the effect of iron therapy alone was made. It should be noted, however, that the dosage of iron used was below the accepted optimal dosage.

Since Stohlman[25] has shown that erythropoietin levels are not significantly increased in animals with mild to moderate iron-deficiency anemia and Kraus[26] has confirmed this fact in humans, the rationale for the combined use of cobalt and iron in iron deficiency appears to be increasingly well established. In the presence of adequate iron, the cobalt-induced increase in circulating erythropoietin would be expected to produce a superior response.

In the group of patients with anemias secondary to other disease etiology, but in whom endocrine dysfunction was not a factor, results were considered to be exceptionally good since all but two showed significant increases in hemoglobin concentration and it is well established that due to failure of utilization, iron is of no corrective value. Tolerance to the medication was especially noteworthy in patients with colitis and gastritis (5018, 4841, 3651).

It was also noteworthy, since this condition is exceedingly resistant to anti-anemia therapy, that four of the group of patients with rheumatoid arthritis (4609, 5229, 1107, 5054) all showed rather striking improvement in peripheral blood values.

In the group of eight patients in whom endocrine dysfunction was associated with the anemia, results were considered unusually good in the four hypothyroid cases since this anemia is known to be exceedingly resistant to treatment.

Summary and Conclusions

Copoietin-ferrous, a new preparation containing an organic cobalt complex and ferrous sulfate, was

used in treating 54 patients with non-macrocytic anemias of varying etiology. Although the dosage of iron was relatively small, our experience has led us to conclude that Copoietin-ferrous is effective in iron-deficiency anemia and that tolerance is significantly improved.

As judged by the response in the "secondary" anemias, the new cobalt compound appears to be fully effective in enhancing the formation of erythropoietin.

Of the 47 patients who were followed to completion of the study, 42 (89.3 percent) showed improvement or complete correction of their anemia after treatment for periods ranging from two to 45 weeks. Only three patients complained of side effects, which were limited to gastrointestinal discomfort in two cases and a mild rash after 119 days of treatment in the third. This rash disappeared promptly upon discontinuance of therapy.

Symptoms of weakness, lassitude, fatigue and irritability appeared to be improved or relieved in the majority of the patients and, generally speaking, patients voluntarily reported an improved sense of well-being as hemoglobin levels increased.

In our opinion, the use of Copoietin-ferrous in the treatment of the common hypochromic and normochromic anemias represents a safe and effective treatment.

1501 Arizona Ave., Suite 5-D.

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Drs. Vance, Black, Pioneer West Texas Physicians, Die

Two outstanding pioneer physicians in West Texas have died in recent weeks.

Dr. James Vance, 84, died in El Paso Dec. 12, 1959.

Dr. Vance came to El Paso in 1905 to do surgery for Dr. Francis W. Gallagher. He was a nephew of the distinguished surgeon, Dr. A. Morgan Vance of Louisville, Ky., and had been graduated from the University of Louisville Medical School in 1899.

He practiced at the old City-County Hospital when it was located near Ft. Bliss. Dr. Vance was the second oldest member of the El Paso County Medical Society and a past president of the organization. He was also Past President of the Southwestern Medical Assn. and was a Fellow of the American College of Surgeons.

Dr. Vance was also an outstanding golfer and hunter. He was six times champion of the Southwestern Golf Assn., and an honorary member of the El Paso Country Club. He was a 32nd degree Scottish Rite Mason.

Dr. W. D. Black, 84, died Jan. 3, 1960, in Pecos.

His long-time friend and neighbor, Dr. Jim Camp of Pecos, recalled the public spirited efforts of Dr. Black, who came to Barstow, Texas, in 1905. The veteran physician was a former mayor of Barstow, where he lived until 1957, and held offices in the City Council, Chamber of Commerce and other organizations. He was a graduate of Baylor Medical School.

A founder of the Ward County Medical Society, he was secretary for 15 years during a time when doctors in that area were few and it was the duty of the secretary to keep the county society organized.

Dr. Black was County Medical Officer for Ward County for 22 years and, after the county seat was moved to Monahans, he served as health officer of Barstow for 40 years. He was also County Health Officer of Loving County for seven years.

Disseminated Coccidioidomycosis Apparently Arrested with Amphotericin B

By Jack A. Bernard, M.D., F.A.C.P., and William M. Tubbs, M.D., *El Paso*

An authority on coccidioidomycosis has pointed out that "the course of acute fulminating disseminated coccidioidomycosis has never been altered by any form of therapy, past or present." Various drugs have been used both clinically and experimentally in the treatment of the disease but evaluation of the drugs has been exceedingly difficult. Ethyl vanillate, stilbamidine and its derivatives have been reported to be of some benefit but the results are not conclusive.

The following is a case report of the use of amphotericin B* in a colored patient with proved disseminated coccidioidomycosis of four months duration with apparent arrest after prolonged intravenous therapy along with aspiration of subcutaneous abscesses with the subsequent instillation of amphotericin B directly into the cavities.

Case Report

A 27-year-old colored male was first seen June 15, 1956, with the history of injurying his right shoulder about three days before. He was thought to have a traumatic bursitis. X-rays revealed no abnormalities. He was given symptomatic therapy but he did not improve and ten days later an orthopedic consultant agreed with the diagnosis of a traumatic bursitis and advised continuation of symptomatic therapy of local physiotheraphy and analgesics.

Three weeks later (July 5, 1956) the patient returned with a swollen ankle. H was given oral steroids for this and improved. Three weeks later (July 30, 1956), he developed an abscess on his chest and an abscess on his right shoulder. He was running a low grade fever of 100 degrees and had lost thirty pounds in weight. The abscess on his shoulder was aspirated and a culture of the pus showed pseudomonas and staph albus. The patient was put on penicillin and streptomycin daily by injection for two weeks. He did not respond and was then put on Albamycin orally. He was becoming progressively worse and continued to lose weight. Daily fever of 100 degrees persisted and new abscesses developed. So on August 22, 1956, he was admitted to Providence Memorial Hospital for further study. An aspiration of the abscess at this time showed coccidioides on direct smear and the diagnosis of disseminated coccidioidomycosis was established (four months after the onset of his first symptom).

Past history revealed that the patient was born in San Antonio, November 10, 1928 and lived there until 1942 at which time he went into the Air Force for two years and was stationed in San Antonio and Alaska. He finally returned to El Paso about two years before he became ill.

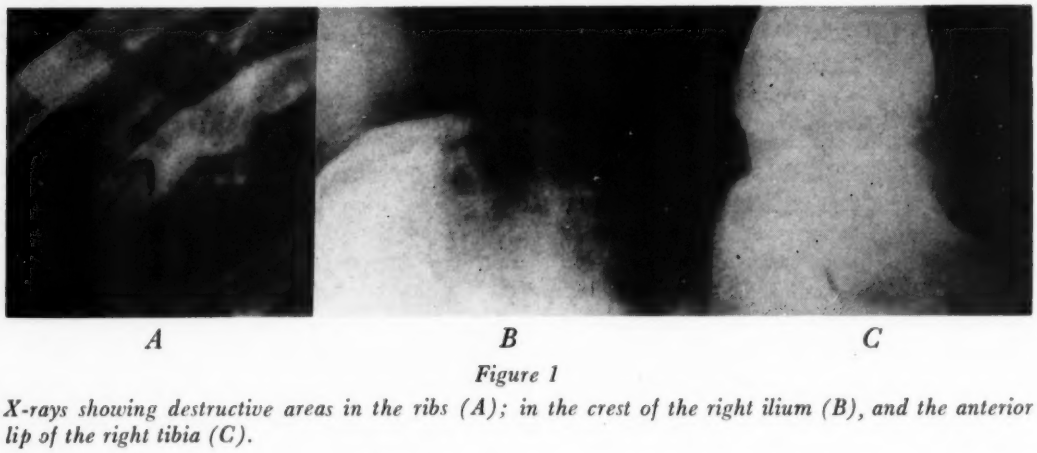
Physical examination upon admission to the hospital revealed a malnourished colored male who appeared chronically ill. Temperature was 100 degrees. He had multiple abscesses of the skin: a large soft fluctuant one in the mid-sternal area, which measured 2.5 centimeters in diameter; a draining abscess of the left wrist; a soft fluctuant abscess on each side of his chest anteriorly just below each nipple each measuring 2.5 centimeters in diameter.

In the right axilla there was a two centimeter gland but there were no other palpable glands. His chest was clear. Abnormal neurological signs were absent and he had no meningeal signs. The remainder of his physical examination was entirely normal except for some tenderness over the right ilium.

Laboratory findings revealed: the red blood count 3.84 million; hemoglobin 12 grams (71 percent); white count 5,900 with 58 percent polymorphonuclears; sedimentation rate was 52 mm/hr. The urine showed a trace of albumin and 3-6 pus cells per high power field on centrifuged specimen. Smear from the material taken from the abscess noted above showed Coccidioides immitis. Serological tests for coccidioidal infection showed a positive complement fixation of 1:256, again confirming the diagnosis of progressive coccidioidal dissemination and indicating a grave prognosis.

X-rays showed multiple destructive areas: (See photographs) there were multiple areas of destruc-

*The trade name of amphotericin B is Fungizone, a product of E. R. Squibb and Company of New Brunswick, New Jersey. Fungizone used in this case was furnished without charge by E. R. Squibb and Co.



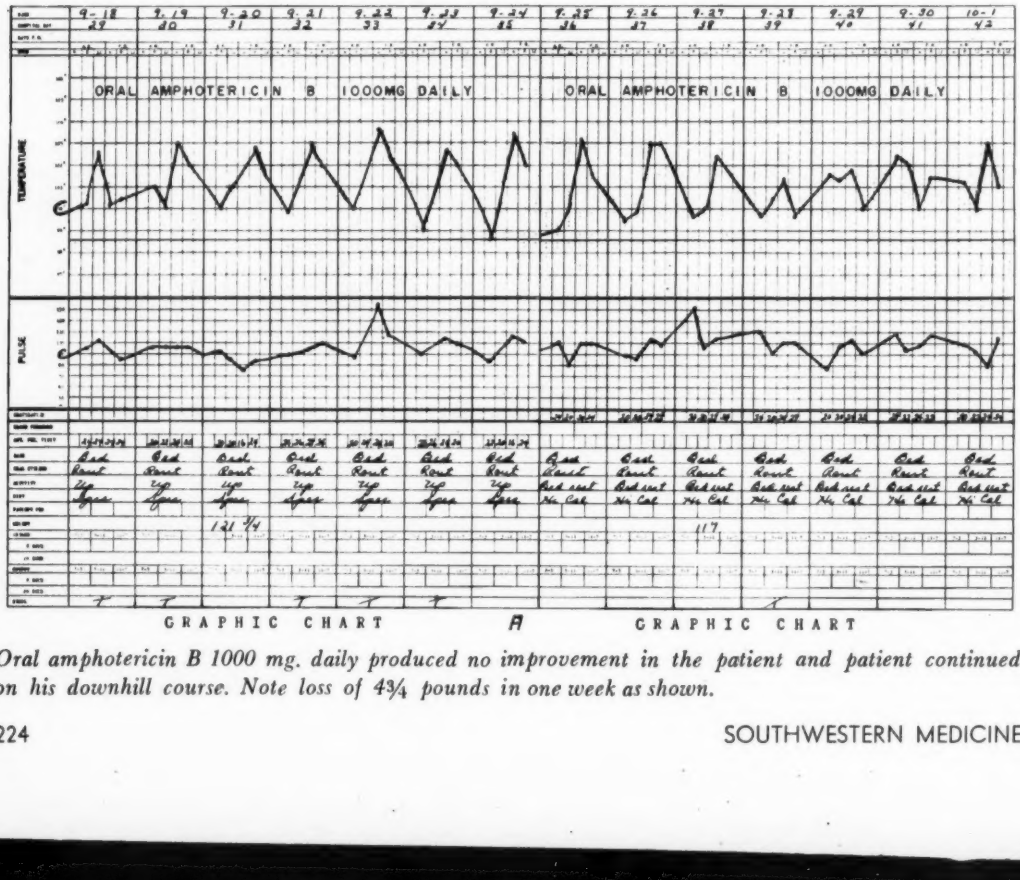
A *B* *C*

Figure 1

X-rays showing destructive areas in the ribs (A); in the crest of the right ilium (B), and the anterior lip of the right tibia (C).

tion of the cortex of the anterior end of the sixth rib on the right and some lesser involvement on other ribs; there was an area of destruction in the anterior lip of the right tibia and a small area of destruction in the right ilium; lower thoracic and lumbar spine x-rays were negative.

Through the co-operation of Dr. Gavin Hildick-Smith, at that time Associate Medical Director of The Squibb Institute for the Medical Research of New Brunswick, New Jersey, patient was started on oral amphotericin B 1000 mgm. daily (four 50 mg. tablets five times daily) on September 18, 1956 and these were continued until October 9, 1956, with no noticeable improvement. (See Graph A) The patient continued to become progressively worse and to go downhill. He was losing weight and spiked a daily fever up to 102 degrees. The disease actually appeared to be more progressive in spite of the oral amphotericin: his fever was becoming higher each day, as high as 103 degrees and 103.6 degrees, and the lesions persisted with the abscess on the shoulder and wrist continuing to



Oral amphotericin B 1000 mg. daily produced no improvement in the patient and patient continued on his downhill course. Note loss of 4¾ pounds in one week as shown.

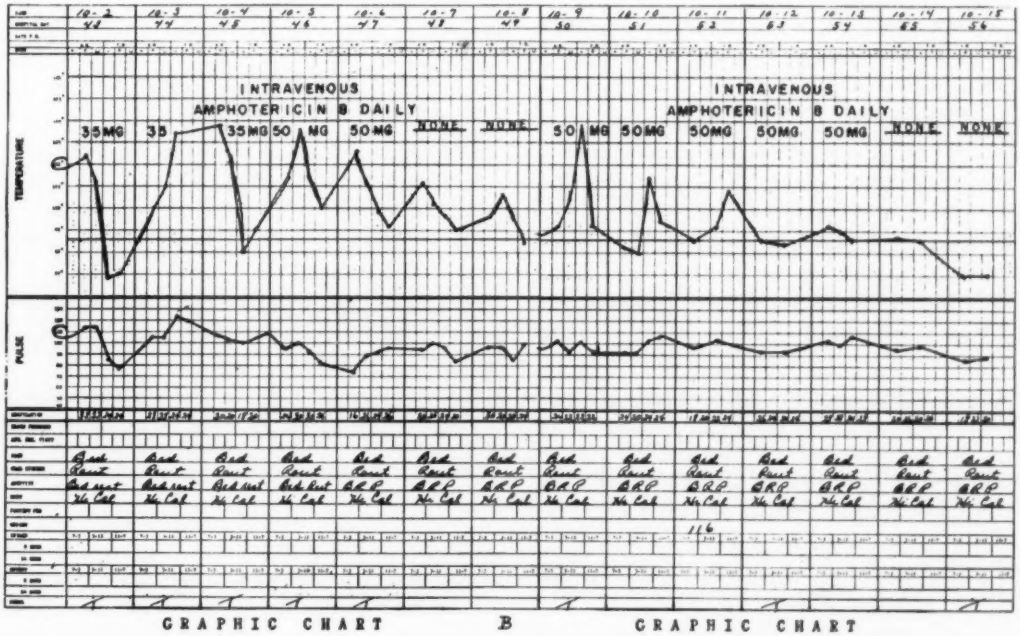
drain. The patient was obviously rapidly succumbing to his disease.

On October 2, 1956, he was started on intravenous amphotericin B therapy. He was given 100 mgm. in 500 cc of 5 percent glucose in water very slowly, but could only take about 150 cc (35mgm.) when he had a chill and his temperature went from 100.6 degrees to 104.6 degrees. He was given symptomatic therapy (aspirin) for this and the next day the same intravenous was repeated, again followed by chills and fever after 150 cc (35 mgm.). Therefore, he was given thereafter 50 mgm. in 250 cc of 5 percent glucose slowly, intravenously, daily five days a week until October 16 (See Graph B) when the injections were decreased to every other day. He was given aspirin for his fever and discomfort and Dramamine orally for nausea. It was felt that it was better to give the intravenous treatments after the patient's evening meal so that his reaction would then occur at night after he had had the entire day to eat his meals.

Liver function studies and urinalyses showed no changes during the amphotericin therapy and BUN was normal after completion of therapy. The only possible side effects were some itching which may or may not have been due to the drug and a slight anemia which developed while patient was under treatment, but patient was anemic before the medication was started.

It was noted that one of the larger abscesses was becoming more fluctuant and it was felt that it would rupture spontaneously so it was decided to aspirate the pus and instill some amphotericin B into the cavity. This was done, using the same concentration and dilution as the intravenous preparation: 50 mgm. in 250 cc of 5 percent glucose, using equivalent amount of this solution as the amount of pus removed. A few days later the abscess was again aspirated and subsequently was aspirated and filled again with the diluted amphotericin. This was repeated every few days until the abscesses had disappeared.

After patient left the hospital, he took the intravenous amphotericin B 50 mgm. in 200 cc of 5 percent glucose on an out-patient basis twice weekly. This was carried out by Dr. U. B. Ogden of Pleasanton, Texas, and patient would go to the office for his treatment following which he would return to his home to have a reaction of severe



Note that the fever and pulse began to subside after 5 days of amphotericin B intravenously, a total of 210 mg. and then he became afebrile after the eighth intravenous injection, a total of 410 mg. of amphotericin B.

headaches, nausea, chills and fever. He would have to go to bed for the remainder of the day, but he would feel well the next morning. He continued to receive 50 mgm. intravenously twice weekly until July 1, 1958. On the days that he didn't receive his intravenous medication he was able to play baseball and felt fine, whereas, on the day he took his treatment he would have to go home to bed. He developed no new lesions. He regained his weight to 140 pounds from the 106 pounds which he weighed when he was ill (his usual weight before his illness was 160 pounds).

On June 27, 1958, patient returned for reevaluation. He had received a total of 5.5 grams of amphotericin B over a 21 month period. He looked well, weight was 135 pounds, blood pressure was 115/90. He showed a large scar over the upper sub-sternal area, a tiny scar over the right shoulder and the left lower chest posteriorly and one on the left wrist. He had some small cervical, small left axillary and small inguinal glands. Remainder of his physical examination was otherwise unchanged.

Laboratory findings were as follows: red blood count 4.58 million; hemoglobin 14.65 grams; white count 7,600 with 60 percent polymorphonuclears; sedimenation rate was 15 mm./hr.; icterus index was 7.2; cephalin flocculation 1+ in 24 hours; BUN was 14.5 mg. percent. Urinalysis was negative with no albumin and no abnormalities in the microscopic specimen.

Radiological studies of the chest, pelvis, ribs and right ankle showed no new areas of involvement and a number of the old previously described areas had healed by sclerosis.

A most important finding was his complement fixation test which was positive 1:64, a reduction of two serial dilutions since his treatment was started in September 1956, which, of course, checked with his clinical improvement.

It was decided to stop his treatment (he had received a total of 5.5 grams of amphotericin B over a 21 month period) and patient returned home and did well, went to school and stated that he felt well except that he got tired easily and that he seemed to run a little fever at times, although he admitted he did not take his temperature.

On November 25, 1959, seven months after cessation of treatment, he returned for an examination. He looked well, weight was 139 pounds, temperature 98.6 degrees. A scar over the sternum and on the upper right shoulder persisted. Physical examination was entirely unchanged. Laboratory findings were again normal. X-rays revealed progressive healing of the bone lesions with no new bone involvement. Complement fixation was positive 1:16, a reduction in titer of another serial dilution during the past eight months during which he received no treatment. (Dr. Charles E. Smith noted that the titer was below generally seen in disseminating disease.)

Finally on January 22, 1960, 19 months after patient's last treatment, he returned for his annual check-up, stating that he had had occasional fever for the last few months at night up to 100 degrees and that he had to quit playing baseball because he would get too tired.

Physical examination revealed no change. His temperature was repeatedly normal. He still had some glands and the scarring as described previously. Laboratory findings were unchanged. Complement fixation test was positive 1:16 which Doctor Smith interpreted as being a stable and reasonable low titer of complement fixation, compatible with his lesion continuing to remain healed and his essentially normal sedimentation rate. He felt that the serology indicated no coccidiodial "activity".

Discussion

This is the seventh reported apparent arrest of a case of disseminated coccidioidomycosis with amphotericin B and one of the first cases in which the drug was used and one of the first cases in which subcutaneous abscesses were aspirated and instilled with amphotericin B, thereby preventing the terrible scarring which commonly results in such cases.

Oral therapy in our patient in the dosage used (1000 mg. daily for two weeks) was ineffective (See Graph A) and actually the patient was progressing downhill rapidly on the oral therapy.

Amphotericin B produced no serious toxic effects in this case other than the side effects of headaches, chills, fever, nausea and vomiting which followed immediately after the intravenous administration of the drug and which were alleviated somewhat by reducing the dosage and by symptomatic therapy with Dramamine and aspirin. Patient was able to tolerate these after effects without too much difficulty and was able to take the medication on an out-patient basis and then

retire to his home to bed and then would be in good condition the next day to carry on normal activity, even to the point of playing baseball.

Further experience will be necessary to establish the dosage and duration of treatment. This patient became afebrile after nine days of intravenous therapy, about 35 to 50 mg. daily, five days per week, approximately 0.4 grams. Thereafter he was given 50 mg. about three times weekly for several months and then 50 mg. twice weekly for a total of 21 months: a total of 5.5 grams of amphotericin B.

To follow the patient one observes his temperature, weight and appetite as well as the laboratory findings of sedimentation rate and serologic studies, the complement fixation titers being most useful in following the course of the illness and particularly as regards prognosis.

Elevation of blood urea may occur if the dosage exceeds 1 to 1.6 mg. per day but this is apparently reversible. Urinalyses were carried out repeatedly in our case but unfortunately blood urea studies were not done during the course of treatment. Repeated blood urea studies at the conclusion of treatment were normal.

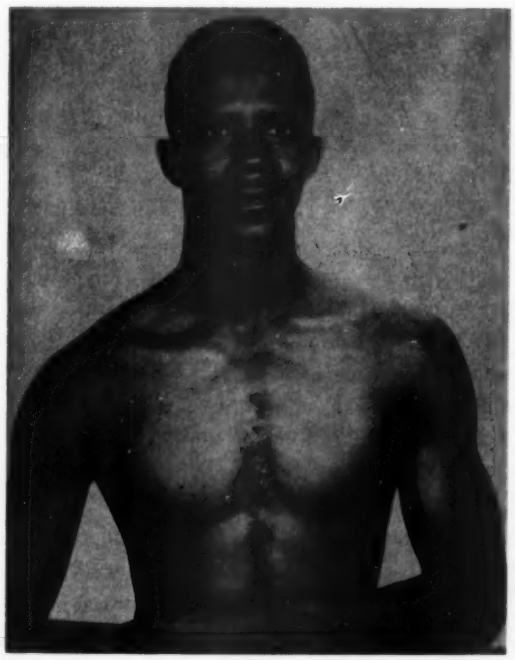


Figure 2

Note scarring over the mid-sternal area, under the left breast and left wrist and the small scar just at the top of the right shoulder where the abscesses ruptured spontaneously; there was also an abscess under the right breast which was treated as described in text and which shows no residual scarring and only slight pigmentation.

Conclusions

An apparent arrest of a case of disseminated coccidioidomycosis in a colored male by the use of intravenous amphotericin B with the injection of the material into subcutaneous abscesses is reported and the lesions so treated healed without scarring. (Figure 2) The drug was fairly well tolerated with no serious side effects. Oral amphotericin B appeared to be ineffective. Toxicity of the drug limits its usage, but it is not prohibitive.

Acknowledgement

Serologic tests were kindly performed and interpeted by Dr. Charles E. Smith, University of California, Berkeley, California. His collaboration was most helpful.

Asknowledgement also to Providence Memorial Hospital and R. E. Thomason General Hospital for their services and co-operation and particularly the radiology department of Providence Memorial Hospital under Drs. Ravel and McVaugh.

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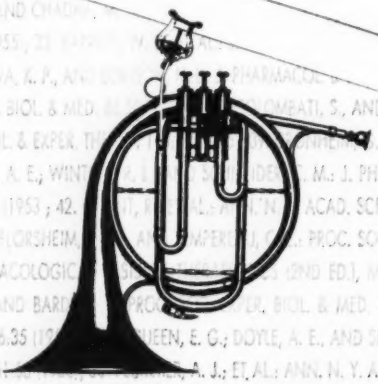

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Clinical Pathological Conference

R. E. Thomason General Hospital, El Paso

Case #1405, February 18, 1960

FREDERICK P. BORNSTEIN, M.D., *Editor*

Presentation of case by IRVIN J. GOLDFARB, M.D.

History: Dr. Nathan Kleban:

On June 27, 1954 the 7th child, a 7 lb. 11 oz. boy, was born to a 27 year old woman and 31 year old man in this hospital. Pregnancy and vaginal delivery were uneventful. Siblings and parents were healthy.

Infectious diarrhea and inflamed buttocks hospitalized the baby from September 22-28, 1954. Weight increased from 9 lbs. 12½ oz. to 10 lbs. 9 oz.

Three weeks before the third admission from February 26 to April 9, 1956 this Latin-American boy developed a cold. Ten days later swelling around the eyes was noted. Several days before he was brought to the hospital the parents detected abdominal swelling and scanty urine of normal color. Appetite remained good. There was a nose bleed on the day of admission.

The child had had no immunization, contagious diseases, or supplemental vitamins. Family history recorded one allergic reaction of unspecified type by the father.

Acutely Ill

The boy was acutely ill, pale, edematous, had a blood pressure of 160/110. In addition to periorbital edema there were blood crusts on the nasal mucosa and ascites. There was no subsequent elevation of blood pressure in the hospital. Response to prednisone therapy for nephrosis was not dramatic.

Repeated admissions to this and another hospital occurred because of upper respiratory infections, chicken pox, ascites which required abdominal paracentesis on several occasions, thrush and failure to do well at home. Nitrogen retention was slight early in the course of the illness, was somewhat more prominent but usually reversible in 1957 and 1958 but progressed to azotemia and uremia in 1959. The second half of the latter year was spent in the hospital.

Last admission to this hospital was on January 29, 1960. The patient was said to have pains in his extremities and had been unable to eat or speak since the day before admission.

Physical Examination:

T. 100.6; P. 82; R. 32; B. P. 110/90; Wt. 41 or 30½ lbs.

Temperature rose to 105.2 on second day and 103.2 on the third, when weights were 29 lbs. 1 oz. and 27 lbs. 2½ oz. Breathing was labored. There was slight periorbital edema. Pallor, distended neck veins, rales, cardiomegaly, hepatomegaly, ascites and neuromuscular irritability were noted.

Hospital Course:

Labored breathing continued. There were liquid yellow and green stools. Twitching and convulsions preceded death on the third day.

Laboratory Findings:

February, 1956—Age 20 months: Blood pressure 160/110; Urea Nitrogen 27.5; Total cholesterol 730, Total protein 4.2, Albumin 2, Globulin 2.2. Urine, S.G. 1.032, albumin 4+, a few white cells, occasional red cells, innumerable hyaline casts, chest X-ray and electrocardiogram normal.

April, 1956—Urine, albumin 2+, to 4+, some red cells, some white cells, a few hyaline casts.

January, 1957—Abdominal X-ray showed ascites due to nephrosis; white count 26,000, blood sodium 130 mEq/L, potassium 3.8, calcium 7.2 mg. percent, fever up to 102.

April, 1957—Albumin 4+, 14 white cells, 4 to 5 granular casts, white count 34,000.

August, 1957—Blood pressure 104/65.

September, 1957—Albumin 4+, 4 to 6 white cells per high power field, 6 to 8 red cells.

January, 1958—white count 22,000, NPN 57, X-ray showed interstitial pneumonitis.

March, 1958—Hotel Dieu—Blood pressure 90/45, NPN 94, Urine, albumin 3+, 5 to 6 red cells, white count 28,000.

May to June, 1958—Urine, S. G. varied between 1.007 and 1.023, albumin 4+, red cells have disappeared, hyaline casts are present, total protein 4, albumin 2, globulin 2.1, Urea Nitrogen 31.5, Sed. rate 51, Total cholesterol 400, CO_2 capacity normal, chlorides 120 mEq/L, sodium 158 mEq/L, potassium 3.7 mEq/L.

August, 1959—White count 22,000, hemoglobin 7 grams, Urine, albumin 4+, 10 to 15 granular casts, packed with white cells, 3 to 4 red cells, Urea Nitrogen 56, CO_2 capacity 22 mEq/L, chlorides 110 mEq/L, potassium 3.6 mEq/L, sodium 140 mEq/L.

July 17, 1959—Chest X-ray—Healthy chest.

July 29, 1959—X-ray abdomen—Findings suggestive of ascites.

Clinical Discussion: Dr. Irvin J. Goldfarb:

All the pediatricians saw this child and most of us were in fairly good agreement clinically, the child suffered of nephrosis—pure lipoid nephrosis. I will just go ahead and discuss the more common renal diseases of children and then include the more exotic diseases with just a word.

First of all, nephrosis, which is what we feel this patient had, is characterized by general edema, proteinuria, hypercholesteremia, and hypoproteinemia, classically without significant hypertension, although hypertension may occur any time in the course of the disease.

Azotemia usually is not present early in the disease although it may be. This disease commonly begins somewhere between 18 months to five years. The course of the disease is insidious, characterized by remissions and exacerbations. The ultimate outcome of the disease is variable, it may have been modified recently by the introduction of the steroid therapy.

Previously it was felt that approximately one-third of these patients died early, about one third of them went into a chronic phase and eventual death and approximately a third of them recovered. These figures may have to be changed upward in terms of cure, although we have not had a long enough follow-up on the steroids to know whether we are altering their ultimate outcome or not.

Etiology Unknown

The etiology of the disease is unknown. Pathologically the kidneys are enlarged. There is thickening of the cortices, presence of liquid material both grossly and miscroscopically. The cells are granular; within degenerated cells, droplets of lipoid material are present. It was thought originally that the disease was confined to the tubule and that the glomerulus was not affected.

However, with present staining, very definitely you can see intercapillary basement membrane changes, fibrinoid degeneration, and for this reason current thought is that it is probably a glomerular disease also, with the changes in the tubule being secondary to the glomerular disease.

Clinically, the disease has an insidious onset, with the child appearing to be well. There occurs gradual onset of anorexia, development of malnutrition, gastro-intestinal disturbances, vomiting or diarrhea, followed by swelling which is usually noticed by the parents. The patients are very susceptible to infection. Pneumococcal disease particularly has always been a hazard, pneumococcal peritonitis, pneumococcal pneumonia, meningitis, and I should mention that superimposed pyelonephritis is not uncommon in these kids. Of clinical signs, of course, edema and oliguria are obvious. I should mention that retinopathy and hypertension is usually absent early in the disease.

Early urinary findings are casts, albumin with minimal or no hematuria. Again, as the disease progresses, a lot of these things can change. Renal function tests, strangely enough, early in the disease are pretty normal, including the B.S.P., as are the more refined tests, the urea clearance, etc.

Studies of the blood show us a reduction in total protein, particularly the albumin; and with long standing disease it may reach the point where you cannot measure the albumin present by chemical means.

Anemia is usually not present early in the disease but universally follows after the patient has had the disease for awhile. Cholesterol and all

other lipids are universally elevated. Cholesterol levels of 600 to 1000 or higher are not unusual. Serium calcium is generally depressed. It isn't shown here on this limited protocol but the serum calcium was 1.8 mEq/L on two or three occasions. Serum sodium, CO_2, may be reduced. If we measure them the amino acid level is also low.

Treatment is not specific, but mostly supportive. Activity—they pretty well limit themselves, so there is no point in giving hard and fast rules on how much activity they should have.

Management

An adequate diet should be given. Salt restriction is probably not necessary, although I certainly would avoid high salt foods. Infections have to be treated early and vigorously. Our case probably began with a very mild upper respiratory infection.

Management of the acute edema—some salt restriction, an adequate protein supply. However, if their potassium is high some think we ought to limit the protein intake. Thoracentesis or paracentesis are indicated if we have interference with respiration.

Treatment

Finally, ACTH or cortisone treatment has changed the picture somewhat. The exact mechanism of action nobody knows. Some people say that it seals up the damage in the capillaries and allows the retention of protein. We know that when the patients respond that their serum protein level will rise and their serum cholesterol will fall. They will stop losing protein in their urine. Their general nutritional status, their anemia, is all corrected with good results.

If you have a failure at one time it may work another time. Steroids certainly are our prime treatment in nephrosis. The follow-up treatment is not one hundred percent agreed on. When diuresis is achieved, most people believe that steroids should be continued, probably in an interrupted form. Three days a week of steroids, to be continued indefinitely, or until the sedimentation rate and the urine has become normal and remains normal.

Recently a new diuretic has been introduced, I have had very limited experience with it, one patient, Aldactone, which is supposed to be an aldosterone inhibitor. It is said to be effective in nephrosis. We do know the aldosterone level is very high in this disease, as it is with refractory heart failure, cirrhosis and a few other diseases which have been refractory to the previous diuretics.

However, even with this diuretic, it is recommended that the patient be started on the steroids first, and in the event of failure, or if you only have limited success, use this strictly to reduce the edema fluid.

Acute Form Rare

In general that covers nephrosis, which again I think this child has. I wanted to mention nephritis, acute glomerulonephritis, or acute hemorrhagic nephritis. The acute form of the disease is rare under age three. This child's first admission was age two, approximately two years of age. We know the etiology of this disease. It is thought to be a hypersensitivity state, usually following an infection with the beta hemolytic strep, and, more specifically, group 12, beta hemolytic strep.

Others assume that it can follow a pneumococcal or staphlycoccal infection, although I don't know what basis they have for this. We do know that even if we don't find the causative lesion we usually have some supportive evidence for a previous strep infection. The antistreptolycin O titer or other similar tests are usually elevated at the time the patient is admitted with acute nephritis.

In many cases at the time of admission the site of the infection is obvious. In an upper respiratory infection you grow alpha beta strep. However, in many, many cases it may follow what may have been a very mild impetigo in which the skin lesions have completely healed; or in which there are only one or two very small lesions, not enough to have made the child ill.

Pathologically, the kidneys are very slightly enlarged, pale, dotted with punctate hemorrhages. The endothelial cells of the glomerular capillaries are swollen. This is considered primarily a capillary disease. Albuminous fluid is found, red cells, white cells, may be found in the space of Woman.

There may be some fibrinous adhesions between the capillary tuft and the capsular wall. The tubular cells are swollen, granular, with hyaline and fatty deposits. However, there is minimal in-

flammatory reaction to the disease, not truly an inflammatory disease. The sub-acute disease varies. Usually there is a large, pale kidney, there is a disappearance of the nephrons, compensatory hypertrophy of other nephrons, there are more capsular adhesions, and epithelial crescents are supposed to be present.

Chronic nephritis is characterized by a small kidney, the capsule is adherent, the surface is finely granular. There is more hyalinization of glomeruli, or these may completely disappear. Many tubules are atrophic, or gone. Extensive scar tissue is found, focal and diffuse infiltrate of lymphocytes is also found.

Now I should mention that nephrosis, or the nephrotic syndrome in, I would say, rare instances, may follow a classic acute nephritis; go on to sub-acute or chronic disease, and end up with a picture which is in no way different from pure nephrosis.

Clinical Story

I don't believe I should go into the entire clinical story of acute nephritis; I will mention the fact that they do put out large amounts of blood, on admission the edema is not too marked but they have some puffiness of eyes, they are putting out albumin, but not in the tremendous amounts the nephrotic patient does, they may have hypertension of severe degree, they may develop cerebral symptoms, or heart failure.

The acute disease is a serious illness and has to be treated vigorously, particularly for the complications. But if you get them through the acute phase of active nephritis, approximately 95 percent will recover, this varies between 95 percent and 98 percent. About 2 percent may go on to chronic disease, and about 2 percent will die in the acute phase. So of the two diseases, acute nephritis carries a much better prognosis than nephrosis, and as a rule again I will say it is not too hard to differentiate these two diseases. There is such a thing as mixed disease, a small number of patients do go on from what you think is an acute nephritis into a nephrotic stage.

The chronic nephritis of course is characterized by periods of general well-being, the kidney changes will go right on. Many of these children will do all right until they reach adolescence at which time they will go on to complete renal shut-down and death in uremia. This is thought to be due to the stresses of the changes of puberty. Those are the primary diagnoses that we have to think about.

Other Diseases

Now for completeness I do want to cover a number of other diseases. Pyelonephritis should very definitely be mentioned, certainly low grade or sub-acute pyelonephritis can be present with a minimum of symptoms which would point to the GU tract.

The patients may not be too ill, there may not be fever, there may not be pain, but repeated infections with repeated insults to the kidney can eventually lead to renal failure, end up with a pretty badly scarred kidney. These children with nephrosis can very definitely have a superimposed pyelonephritis on the original kidney disease which will further contribute to the destruction of renal parenchyma.

Congenital polycystic kidney I will mention. There is no reason why a person with a congenital anomaly of the kidney can't develop any other kidney disease. If this child has it, I think it will be a pathologic finding, I don't believe it is the main thing. I think it is important that we mention that many of the drugs that we are using can cause renal toxicity.

In particular I want to mention Tridione which is used in petit mal and can cause a picture which cannot be distinguished from nephrosis. It has all the classic pictures of a nephrosis but if you withdraw the drug he will get well. If the patient is challenged again with the drug he will again develop nephrosis. Tridione nephrosis does not happen frequently but it can happen often enough and not be recognized.

Other drugs, sulfonamides, have been implicated, and of course we are all acquainted with changes that come with shock, crush syndrome, and with transfusion reactions. Our old friend syphilis is said to be able to cause diseases similar to either nephrosis or nephritis. With amyloid disease, thrombosis of the renal vein, I'm getting in left field now. Finally, nephrosclerosis which is extremely rare in children, can occur with the adult picture of a malignant hypertension.

It has been recorded as young as one or two months, I've seen one in a six months old baby.

This infant developed severe unrelenting hypertension and died within about three or four weeks, but the kidneys were in no way different from the kidney of an adult who dies with malignant hypertension.

Finally, I will just say that I still feel that we are dealing with nephrosis, we may have a superimposed pyelonephritis, and terminally very likely a pericardial pleural effusion, ascites of course we know is present, and cardiomegaly.

Dr. James L. McNeil:

I think I have nothing to add, except that it points out the frustration that you have in trying to treat one of these children, everybody can look at it and everybody can work on it and everybody can treat it and this is what happens to most of them.

Dr. B. K. Byrne:

I had a little advantage in that Dr. Goldfarb brought his stack of charts up to the office last week. The first admission, as I could put it together, looked like nephrosis to all concerned, but I think we are going to find some mixed disease or some glomerular disease in this case because of the calcium. Generally a nephrosis loses calcium, but they only lose the calcium that they are losing bound to their albumin, and they don't get tetany. This calcium got down to 1.8 and the kid did not have tetany.

Clinical Diagnosis: Lipoid Nephrosis.

Dr. Goldfarb's Diagnosis: Nephrosis with possible superimposed pyelonephritis.

Pathological Diagnosis: 1. Chronic glomerulonephritis; 2. Bronchopneumonia; 3. Acute, fibrinous pericarditis.

Pathological Discussion: Dr. Bornstein:

The child was admitted dying with a pneumonia. As all these chronic kidney cases with a nephrotic syndrome, they have pneumonias over and over. This child had a pneumonia, and a uremic pericarditis, and naturally the kidney disease.

The kidneys were extraordinarily small, each of the kidneys weighed less than 50 grams. The surface was fairly uniformly granular, the appearance of both kidneys was the same, so unilateral lesions like a chronic pyelonephritis can be ruled out. The granularity is small and very fine, so you also can exclude a nephrosclerosis. The cut surface showed essentially an atrophic cortex.

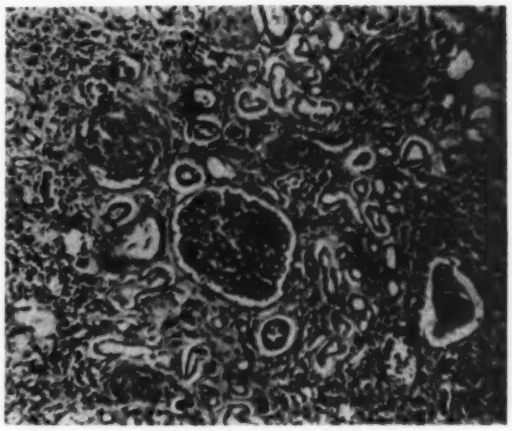

Fig. 1.: Renal cortex, low power, diffuse chronic glomerulonephritis.

Microscopically (Fig. 1) one could see total disorganization of the entire renal pattern. The stroma was infiltrated with inflammatory cells, most of the glomeruli were hyalinized, some had crescents and here and there was a large, swollen glomerulus (Fig. 2). Even the larger glomeruli were involved in active inflammatory disease and were larger than they should be. The tubules were small, atrophic and fat stains were completely negative.

Now we have here a classical picture of a chronic glomerulonephritis. It has been my feeling

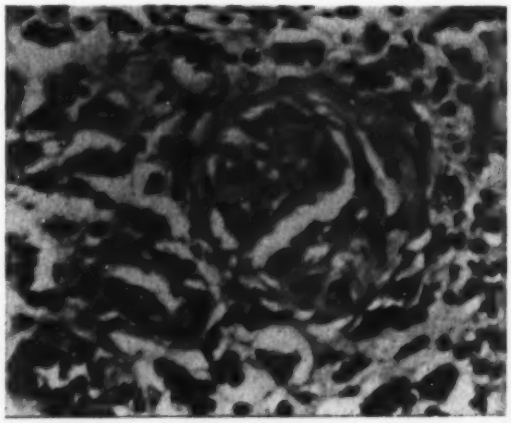

Fig. 2.: Kidney, high power, glomerulus with inflammatory crescent.

in observing chronic glomerulonephritis that this is a disease in which there are always new attacks to destroy more glomeruli. The patient died while an active process went on destroying more glomeruli. We see here beside the destructive of the chronic glomerulonephritis, the superimposed active part.

Now the question then arises, what is the relation of this kidney to the nephrotic stage? When Volhard and Fahr in 1914 started to produce their basic classic, "Description of Various Phases of Renal Disease," they separated the nephrosis from the nephritis. Now that concept has been attacked a great amount, specially by Bell in Minneapolis, and by Allen in his book on the kidney. They state that nephrosis is only a part of nephritis.

My personal feeling and experience is that there do exist cases of pure lipoid nephrosis which clinically are indistinguishable from the nephrotic part of nephritis. I have done one autopsy where I am quite convinced the glomeruli were entirely normal, where there were tremendous amounts of lipoid material in the tubules, where there was tremendous edema, and the patient died of a pneumonia.

If one considers that the pathologist is charged to correlate clinical findings with anatomical lesions, it is very embarrassing to admit that a lesion like a nephrosis can exist independently and obviously has existed here at some time as a part of a nephritic process. I think that this patient started out as a glomerulonephritis, specially because there were constantly red blood cells in the urine and because he was definitely hypertensive at the age of 20 months. There was a nephrotic stage, obvious from the clinical findings. That at death there was none of it left I have adequately demonstrated with the pictures I have shown you.

Dr. Goldfarb:

Fred mentioned the fact that you can't correlate the pathological findings with the clinical story. However, hypertension was only recorded on one admission, it was transitory, the presence of a few red cells is not unusual, with nephrosis. Also, this child had been on steroids for some time and we do know that it has some effect in lowering the total blood fat and I don't know whether it will affect the presence of the lipoid material in the kidneys or not. I just don't know.

Clinically of course you can't tell a chronic glomerulonephritis from a so-called pure lipoid nephrosis, but I think the big weight of evidence is with the onset, with the elevated serum cholesterol, with the massive albuminuria, with the subsequent course, that this fits so-called lipoid nephrosis much more down the line than chronic nephritis. I don't think at any time in the clinical course was the diagnosis of acute glomerulonephritis even considered.

Dr. McNeil:

Pathological findings are always interesting. I think everybody has always, in this group at least, known there are two schools of thought, there is the school that thinks there are separate diseases and there is the school that thinks there are variations of the same disease. There is always the in-between case, which may look like a typical nephritis, and go into the nephrotic stage later on. There have been all kinds of articles written as to whether they are the same or separate diseases.

Regardless of what the pathologist reports, clinically they appear to be, at least in the majority of cases, separate diseases, and certainly glomerulonephritis was considered in this child when he came in. Clinically, there was never any question in anybody's mind of the many who saw him, but what this was a pure and simple case of nephrosis, and all we have to do here is admit the pathologist has some interesting findings. Clinically, it was still nephrosis.

Dr. Bornstein:

This argument really can't be answered except if you really want to raise a question, what do we mean by disease entity?. I am firmly convinced that there is a pure lipoid nephrosis. I am also convinced that chronic glomerulonephritis, or a chronic nephritis, has a nephrotic phase. So there are two different structural disturbances which at one point or another give the same sort of disturbed pathological physiology.

After all, clinical symptoms are expressions of a disturbed physiological state. But I think you should keep in mind, and not simply dismiss the fact, that whatever happened to this child, what this child died with, is a small, contracted kidney that has all the earmarks of a chronic glomerulonephritis.